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DISEASES OF THE NERVOUS SYSTEM

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DISEASES of the NERVOUS SYSTEM

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EDITORIAL COMMENT

Dietary Trends

The body requires adequate food to maintain health or overcome disease. Many factors stand between the acquisition of food and its ultimate utilization by the body tissues. Not the least of these are economic and social, though there are also individual constitutional requirements; but the medical factors predominate.

For years the studies on nutrition were directed toward the chemistry of carbohydrates, proteins, fats, mineral substances and water, which made up the bulk of the diet. An era of dietary restrictions followed, as diet was deemed essential for the proper treatment of physical disorders. The word "diet" itself came to signify a restriction of the food intake. Fortunately a swing occurred in the opposite direction and was probably stimulated by the trends of preventive medicine, that is, the prevention of disease. Far-reaching studies in the field of nutrition have helped to attain a more rational view, and research has been able to either isolate or synthesize new substances which were found to be essential in preserving health.

Conversely, it was demonstrated that their absence caused certain changes in the body structure or function. Whether in the well or in the sick, the trend now is to provide an adequate diet, and where necessary, supplement it by adding vitamin concentrates. Vitamin therapy has been highly publicized, especially by non-medical groups, and some tendency exists to consider it a panacea for ill health.

The Council on Pharmacy and Chemistry of the American Medical Association has repeatedly cautioned physicians against the indiscriminate usage of these substances and urge their use only in recognized deficiency states. The Council has pointed to the doubtful efficacy of multiple vitamin therapy, though in the presence of severe nutritional deficiency, such as starvation, the required individual vitamin preparations should be used in preference to the "shot gun" formulas.

This issue of the Journal contains an article on nutrition in neuropsychiatry by Dr. A. E. Bennett, which illustrates the desirability of adequate food intake in neuropsychiatric patients. Because the mental mechanisms in psychiatric illnesses generally preclude an ideal diet, deficiency states are common complications. Ofttimes a deficiency of certain food elements is the primary cause of the mental illness. It should be emphasized that a deficiency of the vitamin B group, particularly the Nicotinic acid factor, appears to cause acute, or chronic, toxic delirious mental reactions, which some writers have described under the term of subclinical pellagra. While scientific proof of this is lacking, yet the dramatic improvement following administration of the Vitamin B group would indicate that this is an important etiological factor. Although the P P factor and thiamin chloride are the only two vitamins which seem to have a specific therapeutic value in diseases of the nervous system, yet animal experimentation has shown that the lack of other vitamins in the diet may cause changes in the nervous system. A few enthusiastic reports have been published on the use of Vitamin B_6 and a derivative of Vitamin E in neuromuscular disease, but subsequent studies have failed to substantiate these claims. Likewise the value of any of the vitamin preparations in the neuroses and functional psychoses is doubtful except as a tonic or supportive measure.

Further studies in nutrition will doubtless open new fields for therapy, and let us hope that the medical profession will continue the present practice of providing an adequate diet for patients.

Hamilton Ford, M.D.

Iatrogenic Factors in Diseases

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It is accepted as axiomatic that the patient-physician relationship—whether we call it transference or rapport—plays an extremely important part in the handling of the patient's difficulties. One should bear in mind that in this relationship, the partners are not equals. Though concerned with the same problem, they play entirely different roles. The patient looks up to the physician as an authority, accepting cheerfully—as a matter of course—his own relative inferiority. Whatever the affective attitude of the patient toward the physician may be at the first consultation and whatever changes it may undergo in the course of further consultations, that which dominates the patient's attitude is his acceptance, as an indisputable fact, of the superior knowledge of the physician. The physician practicing in areas with a relatively unsophisticated population not infrequently meets with patients who expect him to know all about their illnesses without any further examination than looking at them. In the more civilized urban population, the attitude of patients is essentially the same, with this difference that their respect and confidence go not entirely to the physician, but also to the technical appliances regarded by patients as the necessary parts of a modern medical examination.

On the other hand, the attitude of the physician toward the patient is that of the stronger toward the weaker. Patients whose insight and judgment are within average normal limits, in seeking medical help, not only accept this attitude for granted but welcome it. In this, the physician's rapport with the patient, the affective component plays a very important part. Generally speaking, all other conditions being equal, a warm, sympathetic, genuine interest in the patient's difficulties favorably contributes to the patient's confidence in the physician's diagnosis and treatment. However, it does unfortunately happen now and then that irreproachable, highly qualified behavior of a physician produces entirely unexpected adverse effects on the patient, either directly or through the criticism of the relatives and friends of the patient. A colleague of mine of a very high academic standing and equally high professional reputation, in the role of a consultant on a certain occasion, took great care to carry out a very comprehensive physical examination, which consumed more time than an average, ordinary physical examination. The patient's immediate reaction was that the professor did not feel sure of himself, otherwise, it would not have taken him so long to carry out the examination. In my own experience, my professional reputation was ruined in the eyes of a friend of my patient by a correct diagnosis of which I was very proud. The fatal diagnosis of *Endocarditis lenta* could not be accepted by the female friend of the male patient: ergo, I was no good, notwithstanding the fact that heretofore this woman fully trusted my medical competence. Thus, the physician's rapport with the patient is determined not only by his own attitude, faultless as it may be, but also by circumstances often beyond his control. It appears, therefore, well-nigh impossible to outline, except in general terms, what the proper attitude of the physician should be, inasmuch as the rapport with the patient eventually established depends not on this attitude alone, but equally on the personal make-up of the patient or of other persons involved.

On the other hand, experience shows that in certain instances the physician's behavior, his thinking aloud, prove to be definitely harmful to the patient. It is with such manifestly wrong physician's attitudes that we are essentially concerned here.

Bumke[1] speaks of "iatrogenic diseases," i.e., functional disorders of psychogenic origin in the causation of which the physician himself is to be blamed. He calls attention to the fact that certain remarks made by the physician in the course of taking the anamnesis

evaluating laboratory findings, discussing diagnosis and prognosis are direct etiologic factors in the causation or protraction of various illnesses. He illustrates this statement by factual material, typical of which is the following case:

A recognized authority in internal medicine told a patient of his who had luetic aortitis, but for years got along well: "You should never go out on the street alone. You may collapse any minute." This remark was the cause for fear and preoccupation which made the life of the patient and those close to him miserable for seven years although no "collapse" occurred. Bumke further reminds the reader that in the diagnosis of "heart neurosis" handed out to the patient, what impresses the latter and sticks to his mind is "heart". He becomes thus preoccupied with "heart" and easily finds a substantial proof of his heart illness in palpating the pulse, not recognizing that the rapid pulse is essentially due to worry, anxiety about the "heart".

Bleuler,[2] in calling attention to the iatrogenic origin of certain neurotic manifestations, expresses his experience in the matter in a somewhat sarcastic vein: "The physician solemnly diagnoses 'enlargement of the heart,' whereupon the patient is frightened and breaks down until the x-ray photograph taken by another physician relieves him of his night-mare. The stomach neurotic is not rare to whom a particular food has been forbidden, because of a digestive disturbance "due to a weak stomach," who then in terror consults a string of physicians and has some dish forbidden him by each in turn, until he is at the point of starvation and with or without a physician's assistance makes up his mind to eat what he pleases.

Higier[3] very ably discusses what he rightly calls "pseudoscientific" explanations used by physicians which, in certain psychoneurotics, provoke protracted hypochondriacal complaints. Luria[4] reports clinical observations demonstrating the negative psychotherapy sometimes unwittingly exercised by physicians. The physician's calling the attention of his patient to the hereditary character of his illness; his elaborations on somewhat doubtful findings in the urine, blood, gastric juice, feces, x-ray pictures; casual, metaphorical expressions insignificant to the physician himself, but impressing the patient adversely. Thus, a well experienced and known physician having found achylia in the gastric secretion told his patient, a 36 year old woman: "The cells of your stomach are dead." The patient lost her sleep, appetite and felt very ill, until another physician succeeded in convincing her that her stomach cells were alive. Patients discharged from tuberculosis sanatoria as recovered, live, however, in constant dread, waiting for the third phase of tuberculosis to arrive, because the physician told them laconically that they are in the second phase of tuberculosis. Remarks made by physicians, such as "your arteries are slightly hardened," "your aorta is a little enlarged," "your heart is small," have had ominous effects on certain patients. A renowned internist remarked to his patient: "Instead of a heart, you have a piece of fluttering cheese cloth." This allegorical expression made the patient miserable for 6 years, until MacKenzie destroyed the legend of cheese cloth and prescribed physical exercise including swimming.

Way,[5] in calling attention to "The danger of suggestion in sickness," also cites samples of incautious remarks made by doctors which had nefarious effects: "You ought to have called me sooner"; "You have a very sick child but I will do what I can do for it"; "Go to bed and stay there"; "Be very careful what you do or what you eat".

Hutchinson[6] refers to similar statements made by physicians to patients, such as: A "grumbling appendix," a "slightly raised blood pressure," a "flabby heart," a "suspicious apex," statements which were responsible for the development of nosophobia in certain predisposed patients.

Kanner[7] from his experience in child psychiatry reports cases of more or less marked chronic invalidism in the origin and development of which iatrogenic factors obviously played an important part. Such factors were provided by the physician thinking aloud in the presence of the patient and parents: "It may possibly be the beginning of 'St. Vitus dance,' " "touch" of bronchitis, or pneumonia, or rheumatism; or such remarks as "on the verge of a breakdown," "complete prostration," "nervous," "neurotic," "psychoneurotic," "neuropathic," "psychopathic," "inferiority complex," "mother fixation". As Kanner

points out, whatever meaning these terminologies may have to the physicians who use them, they are accepted by certain mothers as names of actual diseases, which like any other diseases call for particular care, thus paving the way for invalid reactions.

From our own study[8] of records of patients with essentially hypochondriacal complaints,* we gathered material concerned with the general attitude and comments made by physicians, and also with their failure to explain to the patients at the appropriate time the meaning of statements, which though correct, needed clarification. In these patients the physicians' behavior obviously had an unfavorable influence on the course of their difficulties.

Here are a few illustrations:

Case 1

A 13 year old girl complained to her mother of pains in the lower part of the abdomen and of a feeling that she should urinate all the time. The family physician who saw the patient the day of the onset of her complaints let the patient and the mother know his thought that it was "kidney involvement". Three days later he handed out the diagnosis of "streptococcus infection" and advised the patient to stay in bed for 6 weeks. After having been bedridden for about three weeks, the patient began to sigh frequently, saying that it seemed "like something took her breath". At that time the physician told the mother that the girl suffered from a "nervous condition" and banned visitors from seeing her. At the same time, the patient was encouraged by physician and mother to begin to sit up in order to gain strength. She refused because her stomach "hurt so much" that she knew "there must be something wrong". The patient subsequently spent a month, most of the time in bed, in a medical ward of a general hospital. The physical examination and laboratory investigations were negative. Upon returning home she was quite well for a week, then returned to her previous invalid life. It is of interest to note that the patient started to complain of abdominal pains a week before the onset of a new term at school. In her previous school periods the patient often thought how nice it would be to lie in bed all day. She had to get up at about 6 o'clock in the morning and drive 10 miles to school.

Case 2

A single, 40 year old woman, complained of weakness, exhaustion, terrific headaches, neuralgia, neuritis. Her mother added that she suffers everything a mortal has. The patient has been under medical care all her life. The atmosphere of her home was one of continual complaining. At the age of 4½ years she was ill for about 8 weeks with what was called "malignant malaria". She remembers the doctor saying that she would not live. Since then she has been considered delicate. At the age of 30, 4 years after the second operation of uterus suspension for abdominal pain and backache, she was told by her physician that she "would have a real smash up" if she didn't stop her work; she resigned and has held no job since.

Case 3

A married woman, 43 years old, began complaining of heart palpitations and more of g.i. discomfort—belching, heart-burn, jumpy feeling in the stomach—after the birth of her only child when she was 35 and her husband 55, both parents, feeling that it was too great a responsibility at their age to bring up a child. Her abdominal discomfort was recognized as due to spastic colon and she was advised by the physician to have her appendix removed. After the appendectomy, she was treated for a spastic colon with prescriptions which had no good effect either. Then the gynecologist thought that it was "probably a stricture" and gave her cystoscopic treatments. After the third treatment she was taken to the hospital with severe abdominal pain. Again, x-ray showed spastic colon. The physician told her that this was the cause of her troubles, and if the pain didn't clear up all he could see to do was "to make an incision and peep in there and see if we can find anything more that is causing it". About 2 months later she was operated on for the release of adhesions that had formed after the appendectomy. For the next 2½ years the patient was well. Then her boy cut his finger with a penknife. Her old abdominal pain with belching returned. Her surgeon told her, "you seem to get the

* In the Henry Phipps Psychiatric Clinic, Johns Hopkins' Hospital, Baltimore, Maryland

type of adhesions that interfere with your functioning, I guess we will have to operate." After a six-week stay in the hospital, without operation, she returned home and was quite well for a week or so. Then the boy caught a cold. The patient states, "I got all upset after that. The same old pain came back". Back to the hospital she went and was given bromides and colonic irrigations. Her surgeon reproved her, saying, "you don't want to be one of those neurotic people—you never have been—get yourself quiet." She returned home feeling weak, belching and complaining of burning sensations in the left upper abdominal quadrant. This terminated the 8 year period of medical and surgical treatments of a condition recognized as spastic colon.

Case 4

A married woman of 48, whose complaints had centered about the g.i. tract, accumulated in her many visits to doctors various diagnoses: liver troubles, gall bladder disease, dropped stomach, fallen intestines. One physician told her that she was lucky to be on her feet. Her family physician told her he couldn't see how she was able to go around having so much the matter with her. In consequence, she became more and more panicky about her condition.

Case 5

A married man, 35 years old, was treated for Vincent's Angina with Neo-arsphenamine. After four intravenous injections he no longer felt an acid taste in the mouth, gained weight and felt well. But, not having received any explanation by his physician as to the use of Neo-arsphenamine in diseases other than syphilis, he wondered whether he had syphilis. Consequently, within several months he was preoccupied with the idea of syphilis, had ten Wassermanns taken on himself and four on his wife.

Case 6

A married man, 40 years old, has been for 18 years complaining of headaches, dizziness, abdominal distress. Yet, he has been during all these years a very energetic person, an active and successful packing dealer. He made frequent visits to doctors; the physical and laboratory examinations were consistently negative. One of his consultant physicians. on the staff of a well known clinic of excellent reputation, gave him the diagnosis of "brain fag," and "bad nerves," without any further elaboration. Another physician, notwithstanding the entirely negative results of the physical examination, and the obviously hypochondriacal attitude of the patient toward his bodily complaints, told him that he should forget that he ever had a job. The patient recounted: "This was the worst blow I ever had."

The material here presented speaks for itself. The only comment it calls for, on my part, I feel, is the unequivocal statement that it does not contribute anything new, or unfamiliar to the physician practicing any branch of medicine. The only justification for reiteration of obvious facts—even, as Bumke points out, at the risk of becoming unpopular —is that *knowing* is too frequently not followed by *doing:* Notwithstanding the general recognition by physicians of the existence of iatrogenic disorders, caused either by incautious remarks, or otherwise by unwise attitudes of "other physicians," toward their patients or those close to the latter, the fact remains that such disorders are still frequent in hypochondriacally predisposed patients. The very convincing reports in the literature coming from various countries bear testimony to the university of iatrogenic disorders.

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Some of the Neurology of Dementia Praecox*

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Dr. Heldt received his medical degree from the Johns Hopkins School of Medicine in 1916. He volunteered for Army Service in June of 1917, and later went to France with the A.E.F. as Division Psychiatrist for the Eighty First Division. On return to the United States in 1919, he became Clinical Director of U.S.P.H.S. Hospital at Waukesha, Wisconsin. In 1922-'23, he was Psychiatrist-in-Charge of Child Guidance Clinic No. 1 in St. Louis, Missouri, and Norfolk, Virginia. In July of 1923, he came to his present position as Physician-in-Charge, Division of Neuropsychiatry of the Henry Ford Hospital. He is a Past-President of the Michigan Society of Neurology and Psychiatry; a Fellow of the American Psychiatric Association; a Diplomate of the American Board of Psychiatry and Neurology; a member of the American Neurological Association, and several other professional associations. He has contributed a number of articles to scientific journals.

Neurology, in its broadest sense, is that branch of medical science that deals with the nervous system, both in health and in disease. Acceptance of such definition permits liberal consideration of the normal, as well as the abnormal. It is with such thought in mind that it was judged timely, and possibly profitable, to review 100 corroborated cases of dementia praecox that had found their way into a general hospital in the course of the past twenty years.

It is freely admitted that adherence to psychodynamic principles in the study of dementia praecox has been more productive of helpful interpretation than has slavish adherence to the "disease process" hypothesis. Despite this fact, and in the hope that the general hospital setting might somehow add a bit of helpful material, the case records of 339 patients diagnosed as dementia praecox since October 21st, 1920, were scanned, and 100 cases, whose diagnoses were corroborated by institutional care, were selected.

In order to sharpen the insight of the reviewer and to add interest to his quest, it is judged fitting to make reference to a number of previous studies.

The evolution of the dementia praecox concept is succinctly outlined by Meyer.[1] During the past forty years, he himself has added much to such evolution through the formulation of his reaction types based on psychobiological integrations. We must be mindful also of the definition, or delimitation, of the schizophrenic type of reaction, as given by Campbell.[2] The difficulties to be met in any inquiry in the problem of dementia praecox is clearly apparent in the work of Lewis,[3] yet the value of a straightforward review of plain facts and findings is made evident by Dayton,[4] and by Terry and Rennie.[5]

Dunlap[6] was certain that he could not find in dementia praecox a detectable neuropathology. Von Monakow,[7] however, was equally certain that in dementia praecox there was a consistent presence of pathological change in the choroid plexus; in fact, so much so, that Von Monakow[8] was able to make out four distinct types of change. So sure was Von Monakow[9] of his findings that he proceeded to outline a genetic background for the psychoses on the basis of a biologic and a pathobiological interpretation. In this regard, however, it would appear wise to give heed to the strong implications of Gruhle[10] that dementia praecox is probably less a clinical entity than it is a syndrome of variables.

With the advent of the shock therapies, a truly new interest was engendered in the nebulous syndrome of schizophrenia. The insulin therapy of Sakel,[11,12] the metrazol therapy of Von Meduna,[13] the nitrogen anoxia of Himwich,[14] and the electrical shock therapy of Cerletti and Bini,[15] with their various modifications, have done much to focus an increasing interest. The electroencephalograph[16] also has made its contributions.

Bolder still are the surgical approaches. Freeman and Watts,[17] through prefrontal lobotomy, have reported noteworthy changes in psychiatric disorders. More recently, Strecker[18] has applied this surgical procedure to some selected cases of dementia praecox, and feels encouraged by the favorable changes wrought in the behavior of the patient. At this time, too, the partial ablation of Fulton's[19] area 6 by Klemme[20] for selected spastic patients is bringing additional surgical encouragement, and it is probably not far fetched to believe that sooner or later some such extirpation operation will be considered in certain cases of dementia praecox.

Reflecting on the above investigations and

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the work of many other investigators, personal curiosity was aroused to the point where it was judged that it might not be without point to see if the varied and multiple general medical and neurological observations of praecox patients as recorded in records of a general hospital are of more than negative import. Accordingly, 100 case records were carefully selected from a total of 339. The only criterion governing the selection was that the examining psychiatrists be certain of their diagnostic grouping, and that the diagnosis be further corroborated by the patient having spent a period of time in some State or County Institution, and that the physicians in such institutions make a like classification.

For purposes of general orientation, it may be noted that there were 48 males and 52 females, 3 colored and 97 white. The diagnostic grouping is: Dementia praecox simplex 16; hebephrenic 14; catatonic 26; and paranoid 44. The ages and civic status are apparent from Table I. Environment, religion, and occupation are recorded in Table II. Blood counts and blood pressures are noteworthy. The findings are shown in Table III. Observe that hemoglobin percentage is below 80 in 26; white blood cell count over 10,000 in 22; and systolic blood pressure below 110 in 16. The cerebrospinal fluid findings are always of neurological interest. These are found in Table IV, for 60 patients. (Lumbar puncture was not carried out on 40 of the selected patients). Six of the cerebrospinal fluids showed noticeable changes in the gold chloride curves, and two of these fluids showed total protein of over 30 (both a reading of 40). These findings may not be significant in themselves, but are probably truly important in the entire symptom pattern of the patient. Associated somatic morbidities were present in considerable percentage. They are tabulated in Table V, in keeping with the usual loci

TABLE I—AGE AND CIVIC STATUS

Age										*Civic Status*				
Female					*Male*					*Female*			*Male*	
15-19	20-29	30-39	40-49	Over 50	15-19	20-29	30-39	40-49	Over 50	S.	Mar.	Wid.	S.	Mar.
4	18	15	14	1	3	16	20	7	2	32	19	1	40	8
52					48					52			48	

TABLE II—ENVIRONMENT, RELIGION, AND OCCUPATION

Environment			*Religion*			*Occupation*									
Urban	*Suburban*	*Rural*	*Prot.*	*Cath.*	*Hebrew*	*Prof.*	*Housewives*	*Factory Workers*	*Students*	*In Indep. Business*	*Clerks Secys. Salesmen*	*Farmers*	*Maids*	*Others*	*Unemployed*
59	36	5	60	35	5	4	17	21	14	9	12	3	2	2	16
100			100			100									

TABLE III—BLOOD COUNTS AND BLOOD PRESSURES

Blood Counts									*Blood Pressures*					
Hbg. Per Cent			*RBC in millions*			*WBC in thousands*			*Below*	*100-*	*110-*	*120-*	*130-*	*Above*
60-79	80-99	Over 100	2-3	4-5	Over 5	3-6	7-10	Over 10	100	109	119	129	139	140
26	58	12	1	70	24	33	44	22	3	13	23	26	17	11
96			95			99			93					

Hemoglobin determination was not made in the case of 4 patients; no RBC count in 5; no WBC count in 1; and, no blood pressure reading in 7 instances.

TABLE IV—CEREBROSPINAL FLUID FINDINGS (60 Patients)

Cells			*Sugar*				*Total Protein*			
(Number)			*(Mgm. per 100 cc.)*				*(Mgm. per 100 cc.)*			
0	1-4	4-8	60-69	70-79	80-89	Over 90	15-19	20-24	25-30	Over 30
16	36	5	23	29	5	2	9	17	15	2
57			59				43			

Cell count was not obtained in 3 cases; no sugar determination in one; and, no protein determination in 17.

Globulin determinations were negative in all of the 60 cases, as were the Wassermann reactions. Lange (gold chloride) determinations showed slight changes in 6 cases: 0111100000; 0011221110; 0111000000; 0001110000; 0001221000; and 0111100000. These were corroborated respectively by the following mastic determinations: 00000; 32100; 00000; 2100; and 11000. The 60 tabulated cerebrospinal fluids were all free from contamination by red blood cells or blood plasma as far as known.

TABLE V—ASSOCIATED MORBIDITIES (Secondary Somatic Conditions)

Dental Pathol.	*Tonsillar Pathol.*	*Sinuses Infections*	*Impairment of vision*	*Ear Trouble*	*Gastro-Intest. Disorders*	*Genito-Urinary Pathol.*
52	27	15	26	10	28	15

In addition to the tabulated morbidities, 10 patients were afflicted with major conditions of pathology for which they had recently undergone surgery, or were operated upon while in the hospital. These conditions included: Extensive bone graft and fusion; 2 severe head injuries; 2 appendectomies; fracture of left femur; Colles' fracture of right wrist; a series of 7 operations on right foot, including amputation of a toe; a tonsillectomy; and, a thyroidectomy.

for focuses of infection and nidi of pathology. No minor infections or irregularities are included. The dental pathology consists of pyorrhea, caries, abscesses, and extensive gingivitis. Unequivocal chronic tonsillitis was present in 27 and infected sinuses requiring active treatment in 15. Minor errors in refraction were ignored, but 26 patients disclosed impairments of vision necessitating correction. The ear conditions consisted of otitis media, various degrees of deafness and distressing tinnitus. The gastro-intestinal disturbances showed a high percentage of chronic irritable colon with associated constipation in most cases and in some instances diarrhea. Gallbladder, biliary, and gastric disorders totalled 8 in number. The genito-urinary disorders were mostly pelvic conditions and cystitis with 3 cases of prostatitis.

All of these secondary somatic morbidities and pathologies assume importance in relation to the primary psychiatric aberrations through the reduction of the physical resistance of the patient which reduction is usually evident in the total ensemble of the reactions of the patient, and not at all impressive if viewed singly.

The sources from which these patients came are of especial interest because of the general hospital setting, and furthermore, because the 100 cases here reported were all patients well advanced in their schizophrenic symptomatology. Thirty-six reported directly to the New Patient Group, or Receiving Service, and by that Hospital Division were referred to the Division of Neuropsychiatry. Fifty-five came directly to the Division of Neuropsychiatry. However, two spent some time in the Division of General Medicine, two in General Surgery, two in Neurological Surgery, and one each in the Department of Pediatrics and the Division of Metabolism, before they finally came under neuropsychiatric care. Grouped as to, by whom referred, it may be noted that 53 were brought to the Hospital by relatives, 5 were encouraged to come by former patients, 7 were transferred from State and County Institutions, 31 were referred by physicians, and 4 were brought by friends.

Inasmuch as all of these patients had been examined in routine neurological manner by one or more junior physicians and frequently by one or more of the senior physicians, it was hoped that the recorded findings as to pupils, reflexes, ocular fundi, speech, gait, station, and tremors might be especially worthy of review. However, such is not the case, and reliability is not present beyond saying that roughly 10 per cent of the pa-

tients showed pupillary changes that might prove of some significance if opportunity were available for re-examination. The same is true of the osteo-tendon reflexes, and especially is this true of the ophthalmoscopic findings in a like percentage. Each of these points of examination came in for specific neurological comment only in two or three cases, gait and station rarely, and the presence of tremors was noted in only three instances. In cases generally regarded as psychiatric, thoughtfully carried out neurological examinations and reviews are conspicuously few.

That neurological examination and review in psychiatric cases should not be slighted is emphasized by the following case review:

E. M., a married woman of 35, was brought to the hospital by her father because of observed paranoid trends. Her paranoid reactions were clearly apparent, but she was thoroughly cooperative and agreeable to hospital admission for further examination and care. Routine neurological observations and tests by a junior physician were not unusual. Although patient's cerebrospinal fluid Lange (gold chloride) was 0011221110, her mastic 32100, and the total protein 30, with 1 cell and a sugar of 70, along with negative Wassermann reaction, it was judged that these findings might be disregarded since examiners had found no somatic incriminations. She was shortly committed to a State Institution as a case of dementia praecox of the paranoid type, and after three years of treatment there as such, returned to her former civilian environment. She returned to the hospital for examination that must precede her re-enstatement in her rights of citizenship. Neurological examination now disclosed pallor of right nerve head, impaired abdominal reflexes, and equivocal Babinski responses. Scrupulous anamnestic inquiry, with additional neurological examinations by clinic neurologists brought ready agreement that the neurological findings in the case were indicative of a multiple sclerosis that patient had probably been experiencing for some years. The neurologist very naturally asked, "Isn't it possible that the multiple sclerosis is the sole cause of her aberrant condition, including her long stay in a state hospital?" The psychiatrist, however, would insist that the multiple sclerosis was incidental, and that the schizophrenia was too apparent to be anything other than the primary condition. In the majority of such instances, the neurologist remains unconvinced.

In order, also, is a case report disclosing how the neurological impressions and aspects of a case sometimes overshadow a more obvious psychiatric interpretation.

F. B., an unmarried man and a skilled laborer of 33, was admitted to the Division of Neurological Surgery for review. Although the patient's personal statement of his complaint was "My folks think I'm crazy," the examining surgeon, on finding some facial asymmetry, judged that the patient must be looked upon as a "brain tumor suspect." Accordingly, more detailed examinations were undertaken, including an encephalogram. Shortly after that procedure, however, the patient's paranoid formulations came more and more to the fore, and after breaking some windows in attempting to escape his fancied persecutors, the patient was seen in psychiatric consultation, and was judged to be a frank case of dementia praecox of the paranoid type, with rheumatic heart disease, mitral stenosis, and chronic tonsillitis as secondary morbidities. Spinal fluid studies and encephalograms disclosed no obvious pathology. After a further week of observation, the patient was committed to a state institution.

R. G. D. is a young, unmarried man of 22 who, of his own accord on October 6th, 1939, came to the Department of Surgery for attention to a stubborn verrucous growth on the fourth toe of his right foot. The lesion proved to be a congenital arteriovenous aneurysm of the anterior and posterior tibial arteries and associated veins of the right foot, with a complicating fistula. The fourth toe was amputated after previous operation for ligation of anterior and posterior tibial arteries. Seven operations were carried out on the foot between October 25th, 1939, and September 28th, 1940. The patient was first seen in consultation on March 8th, 1940. He came under the full care of the Division of Neuropsychiatry on November 1st, and because of his very evident schizophrenic behavior was transferred to the Psychopathic Division of the City Hospital pending his transfer to a State Institution. Psychiatric studies revealed an individual with average

intellectual assets, but with a rather poorly integrated personality pattern; yet one wonders whether the patient might not have been able to swim abreast of the social stream for a more or less indefinite time had it not been for the apparently necessary surgery in his case. Was the disintegration of his previous social adjustments all due to psychodynamic factors, or were there concomitant attrition factors in his bodily physiology which thus indirectly influenced his stability?

In a general hospital fostering a Division of Neuropsychiatry, experiences resident in the foregoing case reports are not unusual. There is just a little surprise, however, in that such instances should occur in cases as evident as the 100 cases here selected, and all of whom were institutionalized. A cursory scanning of the remaining 229 cases (of the 339 from which the 100 were selected) diagnosed dementia praecox, but not included in this study, shows that the percentage of such experiences is much higher for those cases.

Discussion

This review of 100 selected cases of dementia praecox in a general hospital setting leaves much to be desired; however, it emphasizes the importance of a number of those intangible factors that are so difficult of adequate expression. In the daily contacts with these patients, as their therapeutic and general care must be fitted into the functioning of a general hospital, we constantly see unfolded before us the premises of the psychobiology Meyer was already formulating in 1908 on the basis of "substitutive activity." Clearly apparent and applicable, the tenets of psychobiology are not questioned, and the importance of treating the individual as a whole is recognized. Unity of function and expression of soma and psyche is becoming increasingly acceptable, although there is still too much effort at their divorce and separation. In this latter regard, internists, surgeons, and specialists alike with equal insistence, albeit with a little natural bias, emphasize somatic pathology and disordered physiology, as seen in the elusive changes in vasomotor stability quite as much as the psychodynamic changes reflected in the patient's behavior.

Internist and neurologist frequently ask, What has the whole problem of fatigue to do with this disease you call dementia praecox? It is maintained that an insidious deterioration of the person as a whole is the outstanding manifestation of dementia praecox, and such would appear to be evident in the patient's behavior, but what about the effects of the chronic fatigue process? More than 25 years ago, Dolley[22] judged that he had a reasonable interpretation of fatigue; yet, this very day, we are still asking: What is fatigue? What is its biochemistry? And what is the course of its physiological events? With some of the recent shock therapies, the deterioration of the dementia praecox patient is wiped away, at least temporarily, as completely as is his emotional indifference. With the possible erasure of this deeply ingrained characteristic of schizophrenia comes increasing desire to recast the relative importance of dementia praecox manifestations.

Personally, it is judged that slow strangulation of insight is a somewhat more easily observed change in the patient's behavior than the more inclusive process of deterioration. Gradual asphyxiation of soma and psyche alike over the course of weeks, months, and years, is a point of view that is not foreign to those investigators who believe that anoxemia and cerebral anoxia must come in for liberal consideration when the disturbances of the neurophysiology of schizophrenia are under discussion. Twenty-five years ago, William Koch[23] spoke of the "inherent deficiency in the oxidative process in dementia praecox." Such deduction is receiving considerable support at the hands of a number of investigators at the present time. Looney and Freeman,[24] for example, declare: "Schizophrenic patients do not differ from normal subjects as far as the supply of oxygen to the body tissues is concerned, but show a certain degree of dysfunction in its utilization." A voluminous literature already has been built up about anoxemia and cerebral anoxia, and there are encouraging indications that some progress is being made in deciphering the biochemical minutiae of tissue metabolism, especially brain metabolism. Formerly, much thought was given to the catabolic phases of metabolism. At present, it is judged that arrest of anabolic phases may be of equal concern. Caution and interpretative reserve is fostered by many investigators. Several warnings might well be de-

duced from the contribution by Hartman.[25]

The influence of secondary morbidities or pathologies should always receive full recognition. To just what extent any complicating somatic disorder may influence the psychiatric disturbance is not always clear, and although it may appear incidental, it may, in final analysis, be much more influential. In the case report, F. B., it may be noted that there is a complicating rheumatic heart disease. It is in cases of this kind that Bruetsch[26] would have us consider the possibility of associated cerebral involvement.

Summary

Review of 100 selected cases of corroborated dementia praecox emphasizes not only the genetic dynamic principles of psychiatric interpretations, but also the importance of constantly recognizing the general neurology and special neurological aspects, in the observation, the examination, and the therapy of schizophrenic patients.

Secondary morbidities and pathologies are present in a high percentage of the cases and merit scrupulous review as indicators of more intangible etiological factors.

Review of interpretative literature indicates that speculative neurologizing and psychologizing seem slowly to be gaining an increasing quantum of substantial understanding of the fundamental processes of soma and psyche.

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Toxic Effects of the Common Sedatives

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Toxic delirious reactions (toxic psychoses) are, according to Strecker and Ebaugh,[1] probably the most common abnormal mental reaction seen by the medical profession. Certainly, they are the most common types seen and treated by non-psychiatrists. Toxic delirious reactions include the various deliriums of the acute inflammatory conditions, postoperative delirium, delirium tremens, certain senile psychoses, drug psychoses, and many other types. Delirious reactions are usually dependent upon a state caused by toxins of exogenous or endogenous nature and are essentially self-limiting, in that when these toxins are eliminated the process is usually reversible. However, there is evidence available which shows that the secondary metabolic changes are just as important as the primary toxemia and perhaps more so. These are usually recognized in the late stages, seldom in the prodromal or developing stages, when a change in therapy would prevent the development of the full delirious reactions.

In our work we see many cases in which the common sedatives are either the sole cause or the chief contributing factor to the major abnormal mental reaction presented at the time of examination. Most of these could have been prevented had they been recognized in time. The following cases illustrate the usual course and the symptomatology of these types of mental reaction.

Case No. 1—W. L., a white female, aged 30, came to us with the chief complaints being loss of confidence in herself, worry, fear, nervousness and insomnia. She stated that for two months she had been experiencing a loss of confidence in herself. It was especially noticeable a few days before each menstrual period, at which time she would be afraid to be alone, to go downtown, or ride in a street car. This condition became more severe and she went to a clinic. She was told she had colitis, and was given hypodermic injections of an unknown nature for nine months. She was placed on heavy doses of barbitals two months before her admission to the hospital, and for about three weeks she slept well. She then found that she had to double the dose in order to sleep. Soon, however, she found that this increased dose did not produce sleep and she increased the amount even more. For three weeks prior to admission she was using pentobarbital, amytal, and seconal excessively. These did not relieve the condition at all, but only aggravated it.

At the time of admission, she said she had not slept for four nights. Physical examination revealed a young woman in a moderately agitated condition and showing evidence of loss of sleep. Findings were essentially negative. All drug sedation was stopped immediately, and the patient was given 10 per cent glucose together with Vitamin B_1 and insulin to correct her toxemia and metabolic dysfunction. She was given small doses of insulin as a sedative agent. The patient made slow but steady progress the first week, and during the second week began to sleep quite well. Much of the nervousness, anxiety and fear which she manifested on admission and during the first week largely disappeared. She overcame her insomnia, but visits with her family seemed to upset her greatly so that she began to exhibit more of her agitation and developed a fear of returning home. At the time of her dismissal, however, she was considerably improved and was fairly confident that she could manage at home. She has shown steady improvement since she returned home. She is taking no medication.

Case No. 2—M. L. D., a white man, aged 56, came to the hospital with a complaint of confusion and weakness. He gave a history of a slight stroke 10 years before, but ap-

parently without sequellae. For the past few months he had been less active and more placid in disposition. Three weeks prior to admission he began taking bromides because he was becoming increasingly nervous. From then on he became progressively weaker and walked unsteadily. Ten days before admission his family first noticed some mental confusion. His physician stopped the bromides at once and substituted paraldehyde, but with little beneficial effect. Upon examination, the patient was found to be undernourished and appeared to be anemic. Findings were in keeping with a man of his age. Laboratory tests revealed a normal red cell count, a relatively high white cell count, and a fairly low hemoglobin. Differential count was normal. Blood sugar was within normal limits, but the non-protein nitrogen was above the maximum normal limit. Urine was negative. The glucose tolerance curve was markedly out of line, simulating a diabetic condition. Treatment was instituted, using 10 per cent glucose in saline to correct malnutrition and to hasten removal of the bromide, and insulin in small doses for a sedative effect. He received no other medication. The patient was confused and delusional for four days, after which he became more rational. His recovery was marked by occasional periods of confusion. On his dismissal he was completely rational, although he still was not sleeping soundly. He remained well until his death from apoplexy one and one-half years later.

Case No. 3—L. M., a white female, age 47, was admitted to the hospital with a complaint of nervous spells. She had contracted influenza three weeks prior to admission. Following this illness, the patient developed insomnia and began taking barbiturates. She became increasingly nervous and agitated up to a point where ordinary doses were not sufficient to produce relaxation and sleep, so she increased the dose and frequency of administration. She was taken to a clinic eight days prior to admission at our hospital. Mental symptoms upon admission were about the same as they had been for the past week. She had never had any similar attacks, although she had always been nervous. Family history showed that her father had had one period of insanity and several of her father's relatives were insane. The patient sings frequently at funerals about her home town. At the time of entrance into the hospital, she was in a delirious condition, and was quite delusional, hallucinatory and agitated. Physical examination revealed a thin, emaciated woman. The findings were essentially negative except for a retroverted uterus. Blood counts were normal, as was the differential count. Blood sugar and non-protein nitrogen were both within normal limits. The patient was too agitated to allow a glucose tolerance test to be done upon admission. A tolerance test was done one day prior to dismissal and showed an essentially normal curve. X-rays of skull and chest were negative. The patient continued to be noisy, abusive and delusional for 12 hours, after which she became more rational and cooperative. Twenty-four hours after admission she was completely rational and quite happy. Up to this time she had received no medication or treatment of any kind. Infusions of 10 per cent glucose, were started, and Vitamin B_1 together with small doses of insulin were given to stimulate appetite. Insulin in small doses was given as a sedative. The patient had an uneventful recovery and upon discharge, three weeks later, stated that she felt fine. She has remained well since.

An analysis of these cases indicates that there was a progression of events common to all. Each case had an underlying illness. This was intensified by an increased nervous tension state which produced a subjective feeling of restlessness and nervousness. To combat this, they commenced the use of sedatives upon the advice of their physicians. The desired effect was soon lost and the amount and frequency of the sedatives were increased. A cycle was set up in which a toxic condition resulting from excessive dosage caused more insomnia, and more drug was taken to overcome the sleeplessness.

These three cases illustrate roughly the progression of events in drug toxemias. Case No. 1 came to us in the first state, that is, in the earliest part of the course when the toxemia had developed only to the point of insomnia. The irritability of the toxemia was great enough to overcome the pharmacological sedative effect of the drug that was being taken. The two factors were balancing each other, so that the patient was restless, nervous, emotionally unstable and unable to sleep. The natural tendency of both patient

and physician is to increase the dose on the assumption that the basic condition has become aggravated and that increased doses will produce the desired effect. When this is done, the patient's symptoms progress into the stage of confusion, mild disorientation, dreamlike clouding of consciousness and periods of unreality. The sleep formula is reversed and the patient will doze in the daytime and be awake all night. During the waking periods, the patient is increasingly restless, irritable and begins to manifest increased psychomotor activity. Everyone begins to make valiant efforts to force sleep at night in order to make the cycle conform to past habits, and this increase of dosage or continued dosage forces the patient into an acute delirium or acute delirious mania. The symptomatology here is usually identical to the delirium of acute infections, but is seldom recognized because of the absence of acute infection and the failure to realize that drugs may cause the same symptomatology. At this state, the patient is usually transferred to a psychopathic hospital or ward with a diagnosis of psychosis of undetermined etiology.

Largely through the advertising claims of drug houses, the medical profession has been led to accept the various forms of barbituric acid derivatives as being highly efficient and practically non-toxic. In the last few years, however, more and more investigators have pointed out the potential dangers of excessive use of barbiturates, the need for removing these drugs from the open market and placing them under doctors' prescription. Laymen have been assured unrightfully of the apparent safety of these common sedatives, and through special packaging they can buy these drugs on the open market through identification of the package.

Two factors must be emphasized: (1) that these drugs have a definite pharmacological limit—continuance beyond that limit is definitely harmful to the patient; (2) in cases where there is already a toxic condition present, or where the reaction is severe, sedatives may not control the patient. Through carefully controlled studies, safe therapeutic dosages have been established for all sedative drugs. When it becomes apparent that the accepted dose is not enough to produce sedation in the patient, then no amount of the drug is going to produce the desired results. Over-doses may seem to be producing sedation and hypnosis of a natural type, but the patient is being poisoned. Continued usage and ever increasing amounts of the sedatives will lead to a delirious state far more severe than the original condition which caused the patient to use the sedatives.

The barbiturates are the chief offenders among the common sedatives, but the others are by no means free of producing toxic effects. Several investigators have reported upon toxic manifestations resulting from the long continued use of barbituric acid derivatives. Lambert[2] states that he has yet to find a barbiturate which will not distinctly poison and pervert the personality in some patients. "When a psychosis has once been established through the use of the barbiturates, it takes weeks, if not months, to bring about recovery under the best conditions of institutional care." Bastedo[3] states that, largely through physicians' prescriptions, patients acquire the habit of taking barbiturates regularly, as every night, or two or three times during the day. In a previous report, one of the authors[4] pointed out that this regularity produces a psychological need, in that the patient remembers the effect produced by the drug and when similar conditions occur the patient is irresistibly pulled to the drug in order to relieve the discomfort. That there is actual destruction of brain tissue from chronic barbiturate poisoning has been demonstrated by a number of investigators. Pickworth,[5] who demonstrated experimentally these changes in the central nervous system, reported that: "these changes which occur with any of the drugs of this series consist of the deposition of a homogeneous mucinoid material in the white matter of the central nervous system. The mucinoid material has also been found actually within the nerve cells." Clinical reports, plus pathological findings, definitely indicate that barbiturates in heavy concentrations destroy brain tissue. "One heavy dose of barbiturate does no great amount of damage, but the accumulated damage from repeated heavy doses will eventually destroy enough nervous tissue to lower the efficiency of the patient permanently."[4] This type of phenomenon is chronic and usually more or less irreversible. The toxic delirious reactions reported in the

case histories are acute and reversible. We bring out the possible chronic irreversible end results because of the tendency of the physician to treat toxic delirious reactions of all kinds with barbiturates. By so doing, they add an additional toxin which, if given long enough in doses heavier than the accepted therapeutic amounts, may and will cause irreversible cerebral pathology.

Bromides have long been a favorite sedative among medical practitioners. Their chief use has been as a sedative in hyperesthetic states of the nervous system, although they also are used to relieve pain, quiet reflexes, check convulsions and spasmotic nervous affections.[3] Mortality rate from their use is almost nil, although continued use may bring on a very definite psychotic condition in which the symptoms include restlessness, apprehension, hallucinations and delusions, delirium with disorientation and ideas of persecution. Bromides tend to accumulate in the body fluids replacing the chloride content and are very slowly excreted, so that bromide may be detected in urine weeks after its administration has been stopped. Hanes and Yates[6] have reported that of the total number of patients admitted to their hospital, 16 per cent suffer from bromide psychosis.

Chloral and drugs of the chloral series are commonly-used sedatives. In ordinary doses these drugs, like the others, are relatively safe, but in patients who have a toxic condition already, or in whom the illness is of a very severe nature where they must be used in large amounts, they tend to produce psychic phenomena.

Scopalamine has been used by some to induce sleep. It is most effective where sleep is prevented by motor restlessness. Patients frequently acquire a tolerance to this drug and the dose must be increased. Large doses frequently give rise to confusion, delirium and excitement.[7]

As Robinson[8] points out, the barbiturates are used most widely by persons with mental ill health, either under the direction of a physician, or independently by the patient. In abnormal mental conditions there may be a disturbed metabolism, so that the brain is not capable of utilizing sugar properly. If there is added to this condition a toxic product, such as barbiturates in large amounts, then the ability of the brain to use sugar will be reduced even more as the result of both primary and secondary physiological reactions. When it becomes apparent that there is a state of mental illness developing in the patient, the use of these drugs should be stopped immediately and methods taken to restore the brain to normal functional activity.

Drugs which tend to accumulate in the tissue fluids, such as bromides, will reach a toxic level if use of this form of sedative is continued for long, or if the dose is increased. The drug in such concentration in the body becomes a poison, and lowers the ability of the brain to function normally. The result is a toxic psychosis.

Sedatives give a symptomatic relief. The reappearance of the condition when their effect has worn off indicates that they have no curative action, and continuance of them may increase the intensity of the condition and may start the cycle of events leading to a toxic psychosis.

Summary

Three cases have been reported, illustrating the sequence of events leading to a toxic psychosis when sedative drugs have been taken in excessive amounts over a period of time. There are two important factors to be noted:

1. Drugs have a definite pharmacological limit; continuance beyond that limit is definitely harmful to the patient.

2. In cases where there is already a toxic condition present, or where the reaction is severe, sedatives may not control the patient.

In some patients, sedative drugs seem to have the reverse effect of that expected, and intensify the symptoms for which they have been given. There is a definite progression of events in patients who react abnormally to a sedative drug. To an underlying illness is added the burden of toxemia, further increasing the inability of the patient to sleep. Toxic conditions manifest themselves in the early stages by nervousness, restlessness, insomnia and other evidences of increased nervous tension. These symptoms superimposed upon the original complaints aggravate the total picture, making the use of more and more sedatives necessary, thereby prolonging the vicious cycle.

In cases where it is evident that a sedative

drug does not produce the desired results on the standard dose, the sedatives should be stopped and steps taken to return the body to normal physiological function.

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Parkinsonian Syndrome and Trauma (Cranial and Peripheral)*

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The pathogenesis of Traumatic Parkinsonism is of considerable interest from the standpoint of pathology and from its practical application to medico-legal problems. For many years the symptom-group was viewed only as a functional disorder of coordinate movements without any lesional substratum in the brain. The vast experience in the World War and, especially pathological studies of Von Economo's disease, gave an impetus to a new orientation and a reconsideration of the entire subject. Some facts are presented here in a modest group of cases kept under uninterrupted observation for a period of two years, which may throw some light, firstly, on the relation of trauma to Parkinsonism, particularly with regard to the question of a direct and exclusive effect of trauma on the development of Parkinsonian syndrome; secondly, on the medico-legal angle of the problem, when a decision is to be made on the question of temporary or permanent disability of the victim of an injury. The cases are as follows:

Cranial Group

Case 1—C. O., negro, 30 years old, heretofore in good health, received, during a fight, a violent blow over the head with a poker. He was dazed for a few moments. There was no fracture of skull, as proven by x-rays. For 10 days he was totally disabled, complaining of severe headache. On the 20th day after the injury, he called attention to a tremor of both hands. While there were no neurological symptoms of the sensory-motor apparatus, he nevertheless had some impediment of gait. There was a hypertonia of the four extremities. On the 30th day his attitude in walking, in sitting, and his facies were clearly those of the parkinsonian type. During the following two years, the condition progressed and now the case is typical of Parkinsonism. Attention is called to the fact that, from the time the tremor made its appearance up to the present, the patient complained of excessive dryness in the mouth and of a great desire to sleep.

Case 2—B. H., a man of 41, electrician, in good health, fell off a high ladder while at work, his head striking the ground. He was picked up and carried into a house. There was no loss of consciousness. He complained of severe headache during the following five

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days. The skull was intact as shown on x-ray examination. Ten days later a tremor appeared in the right hand which, from being fine at first, gradually became more and more pronounced. He complained of salivation, which also grew progressively in intensity. The right arm was weak. While there was no paralysis, he nevertheless had difficulty in holding objects. The same diminution of power was observed in the right leg. The reflexes, objective sensibility on both sides, were intact. On examination one could observe hypertonia on the right side. Soon the face began to show signs of alteration in the mimicry. All these symptoms progressed uninterruptedly and the patient at this date presents, in the right arm and leg, as well as in the face, the parkinsonian picture.

Case 3—N. K., 37 years old, laborer, was in good health. While cleaning the windows, he was struck over the head by a lead pipe, which fell from the roof. He was dazed, but there was no loss of consciousness. A scalp wound, which bled considerably, was noticed. X-ray revealed no fracture of the skull. The wound healed on the 15th day. Then he called attention to a weakness in his left leg and a tremor in his left hand. There were no neurological signs of involvement of the sensorimotor apparatus. He complained of a sialorrhea and of a great desire for sleep. The condition remained unaltered. Six months later an examination revealed hypertonia of the left side, a set facies, characteristic tremor of the left hand, Sialorrhea and a tendency to fall asleep.

Case 4—A. D., drug clerk, 39 years of age, in good health, fell down in a dark cellar and struck his head against the brick floor. He suffered no loss of consciousness. There was a scalp wound which healed in five days. No fracture of the skull was present. He complained for ten days of severe frontal headache. He soon called attention to a tremor of his right hand and a stiffness of the entire right upper extremity.

Reexamined three months later, he presented hypertonia of the right leg and arm, difficulty in holding himself straight in walking, a set face with loss of emotional expression. He complained of being flushed frequently and perspiring after each such attack. Neurological examination revealed no signs of involvement of the sensorimotor apparatus.

Case 5—R. Z., male, 29 years old, while skating on ice, collided violently with another man, fell and struck his head against the ice. There was no loss of consciousness. A hematoma appeared over the right parietal region. No fracture was revealed by x-rays. Patient was confined to bed for seven days. A few days later a stiffness developed in the right leg which interfered with his walking. At that time, a coarse tremor appeared in the right hand. Two months later an examination revealed: pronounced tremor of the right hand, hypertonia of both right extremities, stiff attitude of the entire body and a set face with diminished emotional expression. A neurological examination failed to reveal an organic involvement of the sensorimotor apparatus. The condition continued unaltered during the following year. The patient complained of somnolence and marked salivation.

Case 6—L. McC., male, 41 years old, while driving an automobile across a railroad track, was struck by a train. He was thrown out at a considerable distance. There was no loss of consciousness. There were many erosions of the skin on the arms, legs, and thorax, and a marked swelling of the frontal region of the head. No fractures were present. Patient was bedridden for four weeks. He complained of headache. At that time, there was a noticeable tremor of both hands and hypertonia of the muscles of both legs. He expressed difficulty of walking, but no paralysis. In standing or walking, the body leaned forward. Examined one month later, he presented characteristic symptoms of the parkinsonian syndrome, including the expression of the face. During the entire illness, from the time of the accident, there was also a somnolent state with polyuria and polyphagia. A neurological examination failed to reveal organic symptoms referable to the sensorimotor apparatus.

Peripheral Group

Case 1—M. S., laborer, 31 years of age, was thrown out of an automobile during a collision and he fell on his right side. He was slightly dazed. He got up without assistance. Upon examination, the following was noticed: Ecchymosis over the right deltoid region with pain in the arm and shoulder. There was no fracture or dislocation. The pain continued

and a decided weakness was noticed in the entire right upper limb. The movements were greatly limited. This condition remained unaltered during a period of two weeks. At that time, the following additional symptoms were observed: Fine tremor of the right hand which did not disappear on voluntary movements; some stiffness of the muscles of the entire right arm; passive movements of the arm were exceedingly slow; objective sensibility was intact; Biceps and Triceps reflexes were exaggerated. The lower extremities were intact. Three weeks later, it was noticed that the tremor of the hand became more and more pronounced and assumed the picture of a tremor observable in Paralysis Agitans. Two months later the facies of the patient began to change: the mimicry was gradually disappearing and the muscles became set; their contractions during the act of speaking became less and less noticeable. Gradually, the face resembled more and more that of a parkinsonian individual. His speech was also parkinsonian: rapid and in short sentences; the movements of the lips were very slight. In walking, the steps were small, the feet scraping the floor.

The vasomotor and secretary disorders, which were present from the time of the accident, deserve special mention. On the third day a cyanosis appeared on the right arm and on the thorax and upper part of the abdomen, also hyperhidrosis of the right hand. This condition, more than any other, remained unaltered during the entire course of the disease. The patient was seen by me three and six months later. The above somatic condition with the sympathetic phenomena were stationary. It was also observed that the patient was extremely emotional and had brief periods of depression.

Case 2—D. O'C., clerk, 37 years of age, fell down three flights of stairs; he landed on his back, but did not strike his head. There was no loss of consciousness. In attempting to get up without assistance, he felt a sharp pain in the lumbo-sacral region. Upon examination the next day, his physician noticed a discoloration over the left sacral region and extreme tenderness of the entire lumbo-sacral area. The patient could not move during the first five days. X-ray examination failed to reveal any bony or articular lesion. The tenderness of the back gradually subsided, but did not disappear during the following six months. The four limbs were intact. The reflexes and sensibility were not involved. By the third week after the accident the patient was able to walk, but with some difficulty. It was then noticed that the right hand was animated with a tremor; he was unable to hold objects longer than one minute. The tremor was gross. The grip of the same hand was good. Some stiffness was observed in the entire body during walking, sitting down or getting up. Tenderness of the back was present, but not pronounced. In walking he was leaning forward, made short steps, scraping the floor. A month later the expression of the face began to change; it gradually became set; there was no emotional expression. The speech also changed, becoming rapid. A year later, the entire condition appeared accentuated, and the patient presented, in many respects, the characteristics of Parkinsonism. As in the first case, attention is called to the presence of sympathetic phenomena, namely: excessive sweating, sialorrhea, attacks of chills with cardiac palpitation.

Case 3—A. S., chauffeur, 30 years of age, in trying to avoid a truck hit a pole. He was thrown out of his automobile, landing on the left side. There was no loss of consciousness. Besides multiple bruises on the face, there was a large ecchymosis on the left shoulder and excruciating pain in the left wrist. Examined on the fifth day, he presented, in addition to the above symptoms, a stiffness in the left scapulo-humeral joint, a swelling of the left forearm and a marked tremor of the left hand. On the 12th day there was a decided improvement in the surgical condition, but the tremor remained. One month later he presented, in addition to the persistent tremor of the left hand, a drawn face, void of emotional expression, rapid speech and the fixed attitude of the entire body, similar to that of Paralysis Agitans. Like the previous patients, this individual showed sympathetic phenomena: abundant generalized sweating, sialorrhea and cyanosis of the left hand. Six months later the condition was typical of Parkinsonism.

Case 4—N. L., house painter, 40 years old, fell from a ladder to the pavement. He landed on his back. There was no loss of consciousness. He had to be carried into the house.

Examination revealed a marked tenderness of the spine, particularly between the shoulders, an ecchymosis of the left forearm and numerous erosions on both hands and face. The condition gradually improved. On the 20th day, when he was already able to be about, although disabled for work because of some pain in the upper back, a gross tremor was observed in the right hand, so that he had difficulty in feeding himself. The patient's wife called attention to his general mannerism, which was that of a man who is rigid in all his movements. Examined three months later, he presented the typical picture of Parkinsonism of facies, of speech, of gait, and of tremor. There were no symptoms referable to the central nervous system, with regard to reflexes, sensations, pupils and sphincters. All laboratory tests, including x-rays were negative. There were no symptoms of involvement of the sympathetic nervous system. Mental manifestations were present, beginning at the time the parkinsonian symptoms made their appearance, namely: depression anxiety, and outbursts of excitement.

The ten cases of traumatism (six cranial and four peripheral) present the following common features:

1. No loss of consciousness was present in any of the cases.

2. The symptoms, which at first suggested parkinsonism and later proved to be correct, made their appearance in the cranial cases on the 10th, 20th, 15th, 5th, 10th, and 28th day after the injury. In the peripheral cases, the symptoms appeared on the 14th, 21st, 5th, and 20th day after the injury.

3. All the patients, prior to the traumata, were in good health.

4. During the entire period of observation (two years), at no time was there evidence of an organic involvement of the sensorimotor apparatus.

5. In the cranial group, two cases (1st and 6th) presented the parkinsonian manifestations on both sides of the body; three cases (2nd, 4th, and 5th) on one side (right); one case (3rd) on one side (left).

6. In the peripheral group, parkinsonian tremor with hypertonia were on the side of the injury in three cases (1st, 3rd, and 4th). In the case of traumatism to the spine the tremor was also on one side (right).

7. The face was involved in all the cases. The typical attitude of the trunk was present in the entire series of the ten cases.

8. Sympathetic phenomena were observed in five (2nd, 3rd, 4th, 5th, and 6th) cranial cases and in all the four peripheral cases. Of special interest is the fact that these symptoms made their appearance simultaneously with the other characteristic manifestations of the parkinsonian syndrome. While the latter increased progressively in intensity, the former remained unaltered during the entire period of observation.

9. All the patients of both series were of a comparatively young age, in whom the element of arteriosclerosis (as claimed by some writers) could be eliminated.

10. Finally, the question of an infectious element, or of a febrile state immediately preceding the traumatism, or at any time in the patient's life, could be eliminated in every case of the series.

11. Mental manifestations were absent in all the cases except in one of the peripheral group.

12. The cerebrospinal fluid gave negative results in all the cases as to the number of cells and pressure.

13. Finally, each of the cases was totally free from symptoms referable to the sensorimotor apparatus.

Pathogenesis

The close resemblance in the clinical picture of parkinsonism following encephalitis lethargica to that observed in the ten cases following trauma can hardly be considered as a coincidence. At this juncture, particular attention is called to two cases cited by Naville et Morsier[1] in one of which parkinsonism developed four weeks after a fall on the head; in the other case, there was a fall on the left shoulder, which was followed by a persistent weakness of the left arm lasting two years, at the end of which time, a typical picture of paralysis agitans gradually but progressively developed. Both patients were young, but there was also a history of a similar condition in some members of the family, but without a history of trauma. There are also cases in the literature in which cranial or peripheral traumata were followed by symptoms of an infectious state, such as we observe in encephalitis lethargica; namely,

fever, diplopia, somnolence, and which gradually developed into typical postencephalitic parkinsonism. Such cases are reported by Bing.[2] In all similar cases, one is forced to admit that there must have been a latent infection which, through its probable neurotropic character, developed fully after an injury. Stern[3] relates the history of a young woman, who, following a cranial trauma, developed fever with delirium and on the 4th day presented the picture of encephalitis lethargica. Moser[4] reports a similar experience with traumatism, followed by symptoms of parkinsonism. Attention is called to the fact that in all the cases mentioned the gradual development of the parkinsonian syndrome, with or without an infectious state, followed shortly after the trauma.

All traumata are accompanied by a more or less intense emotional or psychic state. There are cases in the literature in which evidences of encephalitis followed later by parkinsonism were present after strong emotional incidents, without trauma or infectious state. Suffice it to mention the 150 cases described by Souques,[5] Van Gehuchten, Guillain, Charcot, Kraft-Ebing and many others. Although opinion is divided with regard to the effect of emotions, nevertheless, the fact remains that a preceding psychic trauma was present in all such cases. As an outstanding example of psychic influence on cerebral circulation, may be mentioned the case of Bachére which was often presented by Charcot at his lectures: The man was very young and in perfect health. From a high ladder he fell down into a basin of water. There was no bodily injury, but the man was unusually frightened. He rapidly developed the parkinsonian syndrome which remained unaltered all his life. When he eventually died, Barré[6] found, on section of the brain, pin-point hemorrhages in the basal ganglia and only in that area.

It is definitely established that parkinsonism following an encephalitis is due to lesions in the hypothalamic region, locus niger, lenticular nucleus, and that cranial traumata in general may be followed by lesions in various segments of the brain, including the mesencephalon, so that pyramidal as well as extrapyramidal manifestations may be present. Some authors observed hypertonia and tremors identical with those of parkinsonism as a consequence of traumatic lesions in the mesencephalon. These observations were made during the World War by Guillain, Barré, Léry, and many others. All such observations lead to an opinion, if not to a conviction, that cranial traumata, even without fracture of the skull, may give place among other lesions (and even without them) to a limited involvement of mesencephalon. One cannot ignore the fact that even in the absence of postmortem verifications, immediately or shortly after trauma, the symptoms of parkinsonism are mathematically identical to those which have developed after an attack of acute encephalitis or other etiological factors and verified at autopsy. The rapidity of the development of parkinsonism shortly after cranial traumata, is certainly strongly in favor of anatomical lesions in the mesencephalon.

As to *peripheral* traumata, it is somewhat difficult to assume such a sharply defined position with regard to an anatomical subsratum. If there still exists a controversy concerning the etiological role of a cranial trauma in development of parkinsonian syndrome, still greater controversial opinions are expressed by many authors in peripheral traumata. At first glance, it seems to be a theoretical impossibility to admit an organic relationship, such as cause and effect, between a peripheral trauma and parkinsonism. Nevertheless, one cannot ignore the strictly observed facts by many reliable authors that peripheral injuries, in which the cranium was not in the least degree disturbed, were followed in a very brief period by unilateral or bilateral parkinsonian manifestations, which never regressed, but, on the contrary, continued to increase in intensity. Such was the case in the four examples of the second group of my series, in which the Syndrome under discussion developed in individuals with no history of other etiological factors. Froment[7] has shown from the physiological angle that "hypertonia and tremor are the result of a disturbance in the sensorimotor reflex, which regulates automatic movements of posture and statics." This proven statement of physiology leads us to understand that a peripheral sensory neurone is just as important in the determination of the extrapyramidal syndrome under discussion as a motor neurone. Otherwise speaking, a disturbance

at any level of this extrapyramidal pathway (afferent or efferent) will produce the same effect. Therefore, a peripheral traumatism as well as a central one may result in parkinsonian syndrome. The succession of the clinical manifestations also favors such a view of the pathogenesis. In all peripheral cases sensory manifestations appear first after traumatism and precede the tremor and hypertonia of Parkinsonian syndrome.

An analysis of many cases of peripheral traumatism, as well as of the four cases reported here, show that vegetative phenomena make their appearance almost immediately after the trauma, and are invariably present during the entire duration of the parkinsonian syndrome. In my series, they were present in the cranial as well as in the peripheral cases. The consensus of opinion is that vegetative centers are frequently involved in a variety of encephalitic disorders through their anatomic connections with the hypothalamic region which is the principal vegetative center. Experimental physiology and neurosurgery have amply demonstrated the influence of mesencephalic lesions on the diancephalon which contains the vegetative centers. In all the cranial and peripheral cases of my series, there were abundant perspiration, sialorrhea or excessive dryness of the mouth and throat, vasomotor disturbances, cardiac palpitation, cyanosis and in some cases somnolence.

The consideration of all the facts mentioned on the previous pages concerning the repercussion of a lesion of one neurone upon another, which is functionately connected with it; finally, the presence of vegetative phenomena indicating the involvement of the vegetative centers almost simultaneously with the extrapyramidal parkinsonian manifestations—all justify the cerebral lesional seat in peripheral traumata similar to that of cranial traumata.

Many years ago Charcot had already expressed the opinion that one cause among others had been ignored by writers on Paralysis Agitans; namely, "irritation of peripheral nerves following an injury."[8] It is therefore indicated that in the consideration of peripheral traumata in relation to parkinsonism three pathogenetic elements are to be borne in mind; namely: (1) the seriousness of an injury to a peripheral neurone, which causes a disturbance in the sensorimotor reflex, regulating automatic movement of posture; (2) the presence of vegetative manifestations which point to the central seat of the disorder; (3) the influence of strong emotional shock by recalling the case Brachére autopsied by Barré (see above).

Summary and Conclusions

Six cases of cranial trauma and four cases of peripheral trauma without loss of consciousness are described here in relation to Parkinsonian Syndrome.

Arguments are presented in favor of a direct relationship as cause and effect.

Clinical and anatomo-pathological data are cited to corroborate the above contention.

The chronological development of some of the parkinsonian manifestations shortly and even very shortly after the trauma and the uninterrupted progress of the disease, speak strongly in favor of trauma as being an immediate etiological factor in Parkinsonism.

Trauma, whether cephalic or peripheral, may produce small hemorrhagic foci in the extrapyramidal area and develop the clinical picture of Parkinsonism observable in late stages of Encephalitis lethargica.

That the mesencephalon is involved, can be seen from the presence of vegetative symptoms, the center of which lies in the mesencephalic segment of the brain.

The value of trauma as a causative factor in Parkinsonism has an important practical application in Medico-legal practice, when the question of partial or total disability is presented for a decision.

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Nutrition in Neuropsychiatry

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Deficiency factors are becoming more firmly established as important causes in many neuropsychiatric disorders.

In organic neurology, pernicious anemia and pellagra are classic examples of dietary deficiencies which cause combined system degeneration. The specific action of liver extract relieves pernicious anemia. Even advanced neurologic manifestations of this disease are modified by persistent therapy keeping the red blood count above 5,000,000. It has also been shown that the disease can be adequately controlled by large doses of brewers yeast. Some part of the vitamin B complex carries the specific anti-anemic factor of this disease. The degenerative changes in the lateral and posterior column in pellagra respond to the specific action of nicotinic acid. Others of the B complex are undoubtedly causative factors in pellagra, and nicotinic acid therapy must be followed up with other dietary treatment to be curative.

Our concept of neuritis is undergoing revision in the light of newer information about avitaminotic disease. Some authorities recommend the term neuropathy for neuritis in order to eliminate the concept of inflammation as a cause. It is not proved that vitamin deficiency is always the etiologic factor and thiamin deficiency is not the sole vitamin at fault. In many cases, gastric and hepatic disturbances are present, as in alcoholism, pernicious vomiting of pregnancy, intestinal disturbances in children with metallic poisoning, starvation cachexia, and diabetes. Diabetes usually prevents assimilation of accessory food factors, thus leading to vitamin B deficiency and resultant peripheral nerve degeneration.

We now have a more effective therapy for the neuritides. A high vitamin, 4500 calorie diet, reinforced with thiamin, 10-50 mg. per day, is indicated. In many cases this is not sufficient to relieve neuritic pain and other B complex factors must be supplied in the form of brewers yeast, liver extract, vitamins B_6 or E.

In our work with artificial fever we have found patients whose vitamin reserve becomes depleted; unless it is protected a polyneuritis will develop. We use large doses of thiamin routinely between heatings to prevent this complication.

Acute hemorrhagic polioencephalitis of Wernicke, seen in the course of chronic alcoholism, has been shown to be a vitamin deficiency disease.

In spinal cord atrophies and muscle dystrophies, the possibility of a nutritional or starvation factor as a cause has long been suspected. Recent experimental and clinical evidence suggested that vitamin B_6 and E may be deficient in patients suffering from amyotrophic muscular dystrophies. At least temporary improvement occurred in some severe cases of amyotrophic lateral sclerosis after administration of large doses of wheat germ oil and the tocopherols. Unfortunately the follow-up studies by most observers have been disappointing. The probabilities are that neither vitamin B_6 or E deficiency are causative factors in either muscular dystrophy or spinal cord atrophy.

Much re-evaluation of reports in the literature of vitamin therapy in neurology is necessary. Especially over-enthusiastic are the reports upon thiamin for relief of pain, neuralgias, and chronic progressive neurologic diseases. Further evidence is necessary also to evaluate reports of successful treatment of paralysis agitans by Vitamin B_6.

Psychiatric therapy has been greatly advanced of late years by better knowledge of nutritional disorders. The discovery of nicotinic acid deficiency as a cause of black tongue in dogs and its similarity to human

pellagra is a fundamental contribution worthy of the Nobel prize in medicine.

Acute nicotinic acid deficiency is a positive causal factor in acute alcoholic delirium, Korsakoff's psychosis, pellagrous psychoses and in many senile confusional stuporous states. Undoubtedly other nutritional factors and deficiency in the B complex are present in these disorders. The fact remains, however, that adequate nicotinic acid therapy is life saving and curative. Our present knowledge is sufficient to prevent the pellagrous neuropsychiatric diseases.

Undernutrition in psychiatric patients is frequently an important therapeutic problem. For a number of years prior to the advent of insulin shock treatment, I had used insulin in non-shock dosage for weight gaining purposes. Undernourished patients uniformly gain 3 to 4 pounds a week if given a high caloric diet and a daily dose of 15-25 U. of insulin. This treatment makes the psychiatric nursing problem easier, tends to make the patient more accessible and shortens convalescence. Usually, improvement in mental reaction coincides with the physical gain. Since the introduction of insulin shock therapy we have continued to be interested in the metabolic effect of the insulin treatment. In cases of good recovery the average weight gain has been 13 pounds. Those patients not showing a weight gain have usually not responded well. Furthermore, while the immediate results of insulin shock treatment have been superior to the former non-shock, simple weight gaining regime, the sustained results from insulin shock have been little if any better than from the previous therapy.

C. Richter and P. Young recently have shown that rats, when permitted free access to various food elements important to the diet, instinctively select food containing needed vitamins. The animals take as much or as little as they want of each type of food. The tongue and taste buds regulate their diet. When nerve connections to the tongue are cut the animals lose the discriminatory ability and become sick. Animals with deficiency of fats prefer fats; those deficient in vitamin B pick foods containing the needed vitamin, etc. In other words, the animals, when able, will pick a diet better for them than that worked out by nutrition experts. Humans seem to have largely lost these health saving conditioned reflexes.

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Book Reviews

PSYCHIATRY FOR THE CURIOUS. By George H. Preston, M.D. Illustrated with sketches by the author. New York and Toronto. Farrar and Rinehart, Inc., 1940. Cloth, pp. 148, Price $2.00.

This book, unlike other books written to give the layman an appreciation of what psychiatry is, accomplishes its purpose. In a simple, easy conversational fashion, the author presents interestingly, informatively and understandably the principles upon which modern psychiatry is based. Theory and example are sound and lucidly explained. Sensationalism is avoided and technical language, when used, is adequately defined. The book stimulates, informs and satisfies the reader without imbuing him with an over-enthusiasm to classify, diagnose and otherwise maltreat his fellows. Instead, the simplicity of the book gives the reader a sound and respectful appreciation and understanding of human behavior, of the influence of the environment upon the individual, of the formation and development of behavior patterns, and of the way in which emotional attitudes control and direct conduct in every life situation.

Each of the various types of mental disorder is skillfully discussed in charmingly commonplace language. The reader is made to realize that the psychoneurotic patient is ill with something real and painful to him, however imaginary to other people; that the paranoid patient can be understood in terms of the man who stumbles awkwardly over the cat and then reacts, not by kicking himself for his own awkwardness and inattentiveness, but by kicking the cat instead; that delusions, hallucinations, phobias, projection mechanisms and persecutory reactions can all be understood in terms of every day behavior carried to an extreme, and hence are not mysteriously inexplicable manifestations to be regarded with fear and hopelessness.

Although written for the layman, the medical man, including the psychiatrist, can well use this book to make clear to himself and to his patients the psychiatric problems that are to be met in private practice.

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PRINCIPAL NERVOUS PATHWAYS, 2nd Edition, by A. T. Rasmussen, Ph.D., The MacMillan Company, New York, 1941, Price $2.50.

The second edition of this deservedly popular book is not essentially different from the first. The number of pages and plates and the arrangement of material have been retained without alteration. Changes occurring in the text and diagrams of the new edition have been limited to those made necessary by advances in this field during the past ten years. New information on the exact location of the conducting pathways, particularly the spinothalamic tracts, and more precise subdivision and naming of the thalamic nuclei are the most important and necessary alterations that have been made. Rasmussen's book should be in the hands of every medical student during his courses in anatomy and physiology of the nervous system. Careful study of this small volume during this period will give lasting pictures of the functional organization of the nervous system with the minimum of effort. To the busy clinician Dr. Rasmussen's Work should be an easily available source of reference.

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THE CARE OF THE AGED. (Geriatrics.) Malford W. Thewlis, M.D., The C. V. Mosby Company, St. Louis, 1941.

This is the completely rewritten, third edition of the textbook on Geriatrics, which appeared in 1919. The only earlier work on the same subject was Nascher's book in 1914.

Thewlis' pioneering effort in the problem of the care of the aged has been well established. Throughout the book, the author demonstrates his deep sympathetic interest in this special field, and he frequently shows a frank desire to place his own vast knowledge of the subject at the service of the general practitioner.

The book is divided into five sections. The first section deals exclusively with the general aspects of senescence. A brief history of Geriatrics is followed by a discussion of the anatomic and physiologic changes in old age, both in health and disease. The final chapters include prolongation of life, hygiene, and economic as well as medicolegal problems of the aged. Section II is devoted to preclinical medicine and the remaining sections to the various infectious and noninfectious diseases.

The author has made a successful attempt to cover every field of medicine in the volume. The book is well written, though there is considerable repetition in many of the chapters. The case reports are interesting and well done descriptively. The numerous tables, illustrations and photographs have been carefully selected. Full reference to source of material together with an excellent bibliography make this book of special value.

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MAN, The Mechanical Misfit—by G. H. Estabrooks—The MacMillan Company, 1941—251 pp.

Professor Estabrooks in this book has provided the lay person with a light but scientific exposition of evolutionary development of life on the earth, and has set forth his thesis that man is a mechanical misfit, will eventually disappear and will be succeeded by another form of animal life, thus repeating the past. The book is easy reading and will yet give the reader more than mere information for drawing room conversation. The author in the introduction apologizes for faults of omission and commission. He has not intended to give minute detail or more than general chronological order. I think it important to combine the broad aspects of the story of evolution in one readable book and in this he has well succeeded. I have no especial quarrel with him about the content. In general I agree with his thesis. I should prefer that he had used the term psychosis instead of insanity, hospital instead of asylum, and think that his sole use of the term psychiatrist need not have been explained as meaning "nerve specialist," which latter a psychiatrist exactly is not. I think also that the general paretic these days is not "generally insane for six months before anyone realizes it," although doubtless exceptionally true. Certainly both doctors of medicine and the lay public have become more alert to the

recognition of early mental disorder. These are minor criticisms, compared with the value of the book as educational lay reading and for use in teaching nurses and medical students.

Glenn Myers, M.D.
Los Angeles, Calif.

LECTURES ON CONDITIONED REFLEXES, Volume II, CONDITIONED REFLEXES AND PSYCHIATRY by Ivan Petrovitch Pavlov. Translated and Edited by W. Horsley Gantt, M.D., B.Sc. International Publishers, 381 4th Avenue, New York, 1941, Pp. 199, Price $4.00.

Pavlov, the great Russian physiologist, in the later years of his life, became interested in psychiatry. Quite early in his work with dogs, Pavlov observed that they vary a great deal in their conditioning. He classified his dogs as those in whom the excitatory phases predominated, and another group in whom the inhibitory phase was more developed. He also noticed that when both positive and negative stimuli were applied, the dogs became confused, and did not know which way to turn. In other words, he was able to produce an experimental neurosis. This stimulated Pavlov to attempt to apply his method to the study of mental diseases. The present volume is an ingenious attempt to explain certain symptoms by the disturbed conditioning of the individual in connection with a weak nervous system. There is no question that some of his ideas are quite correct. Thus, in the study of anxiety states in children, we know at the present time that inconsistency of attitude on the part of the parents, when at times they are very severe, and at times over indulgent, contributes a great deal toward the emotional instability of the child.

Pavlov's views on schizophrenia are of especial interest. He believes that, given a somewhat weak nervous system and a large number of strong stimuli, the individual, the same way as the animal, responds by complete inhibition resulting in a condition resembling catatonic stupor or a complete withdrawal from reality. Of course, there is great danger in trusting too much to an analogy between animals and human beings, in whom speech is a more important factor than it is in animals.

The volume on the whole is extremely stimulating and points a new way of investigation in the field of psychiatry. We ought to be very grateful to W. Horsley Gantt of Johns Hopkins University, one of Pavlov's students, for this translation. There is an extremely interesting farewell address to the youth, in which Pavlov embodies his ideas about research, which is worthwhile reading over and over again.

J. Kasanin, M.D.
San Francisco, Calif.

THE PSYCHODYNAMICS OF ABNORMAL BEHAVIOR. J. F. Brown, with the collaboration of Karl A. Menninger, M.D. McGraw-Bill Book Company, Inc. 1940. Pp. 484. Price. $3.50.

This is one of the best books on abnormal psychology which has been published in the last twenty years. The author, who has an unusual background in academic psychology, Gestalt psychology and psychoanalysis, presents the complicated concepts formed by these disciplines in a clean, well thought out manner.

In the first part the author deals with nature vis. nurture with discussion of what is normal and what is abnormal, and clarifies the relationship of mind and body as expressed in the new field of psychosomatic medicine. The second part deals with symptoms as an expression of disturbances of emotion, will, adn intellect. It corresponds largely to physical diagnosis which a medical student must study before he takes up the various diseases. The third chapter is by far the most illuminating in the book, and present a very clear picture of our modern ideas in relation to personality. It is entirely a dynamic presentation and draws a good deal on psychoanalysis to explain the various drives and forces operated in the formation of personality. The last two chapters deal with mental diseases and the function of psychiatry.

The reviewer has used this book as a text in teaching junior medical students and in his opinion, it is by far the best textbook of psychopathology we have at the present time.

J. Kasanin, M.D.
San Francisco, California

ABSTRACTS

Title: THE GINGIVAL CHANGES PRODUCED BY DILANTIN SODIUM

Leon J. Robinson, M. D.
Monson State Hospital, Palmer, Mass.

A brief review of the gingival findings by other authors and the lack of confirmation of vitamin C deficiency was presented. In the present study, it was found that dilantin sodium produced gingival changes to a marked degree in 5%, and to a mild degree in 19%, of epileptic persons receiving dilantin sodium as an anti-convulsant. The gingival changes more frequently occurred in younger persons, proved to be a local reaction unaccompanied by constitutional effects, and when present was found to occur only in gingival areas where teeth were present and where no other gingival pathology such as pyorrhea alveolaris existed. The progressive changes in the gum were graded according to their severity into five stages, clinically described as follows: Stages: 1st, redness, 2nd, elevation, 3rd, elevation, 4th, overgrowth easily traumatized, 5th, interference with chewing. Earliest onset was two weeks after medication was started and might appear anytime thereafter. There was no relation between the gum changes and the dosage of dilantin sodium nor the sex. The gingival changes were not influenced by the administration of ascorbic acid 450 milligrams daily. Because of Ziskin's favorable report on estrogenic therapy in some forms of gingivitis, in our series we administered progynon D H (Schering) orally, using 600 units daily for three months without noting any beneficial effect. The only effective means of insuring regression of gum changes consisted in the withdrawal of dilantin sodium. The fact that gingival changes occur did not demand omission of dilantin sodium provided the gums did not progress beyond the third stage, provided examination was made at 2-4 week intervals, and provided of course the dilantin sodium produced a satisfactory anticonvulsant effect.

Title: DOUBLE SYMMETRICAL PORENCEPHALIES (SCHIZENCEPHALIES)

Paul I. Yakovlev, M.D., Waverley, Massachusetts and Richard C. Wadsworth, M.D., Waltham, Massachusetts

There is a distinct morphologic and histologic difference between the porencephalic defects in the cerebral mantles arising as a result of destructive lesions with necrotic softening and those arising as a result of arrest of development in the walls of the secondary cerebral vesicles. The latter are true clefts in the wall of the hemispheres and are connected with the ependyma of the ventricle by a pia-ependymal seam. These clefts tend to develop along the zones of cleavage of the primary fissures of the cerebral mantle. If such a defect is associated with a severe hydrocephalus, the wall of the cerebral vesicle in the region of the defect becomes stretched out and the lips of the cleft separate along the seam. The cleft becomes a wide gap in the cerebral wall. Five cases are presented illustrating degrees of transition from narrow clefts to wide gaps amounting to a symmetrical agenesis of almost the entire convexity of the cerebral hemispheres. Such defects may be called "schizencephalies." They are invariably associated with widespread malformations in the cerebral mantles such as microgyria, heterotopias, monster nerve cells, binucleated nerve cells and islands of spongioblastic tissue scattered in the subarachnoid space. (Lantern slides.)

Title: RESEARCH IN MENTAL DEFICIENCY WITH SPECIAL REFERENCE TO BIRTH INJURY

Clemens E. Benda, M.D.
Wrentham State School, Wrentham, Mass.

After discussing some general outlines of research in mental deficiency, the problem of the sequela of birth injuries was presented. After excluding cases of mongolism, a number of fifty autopsies have been studied. Nine were familial morons or imbeciles without neurological signs, and with no history of unusual birth. Ten cases were cases of developmental disorder before birth or post natal abnormalities without relationship to birth. Of the remaining thirty-one cases, thirteen had a history of difficulties at birth; while in eighteen instances, no unusual factor was mentioned. From those eighteen cases without a history of birth injury, one case of cystic brain degeneration was suggestive of a pathology which indicated asphyxiation. From those thirteen cases with a positive history, three cases showed cystic degeneration of the white matter probably due to asphyxiation; and four cases showed cortical atrophy, thickening of the meninges, remnants from old hemorrhages and alterations which were consistent with a birth injury but no proof of it.

Newsnotes

Thomas W. Salmon Lectures—1941

"Psychoneurosis in Peace and War and the Future of Human Relationships"

By Dr. R. D. Gillespie

Under the general sponsorship of the Salmon Committee on Psychiatry and Mental Hygiene, appointed by the Council of The New York Academy of Medicine.

Final dates for the Salmon Memorial Lectures which Dr. Robert D. Gillespie, psychiatric specialist of the British Royal Air Force will deliver in key cities of this country and Canada, have been announced by Dr. C. Charles Burlingame, chairman of the Salmon Committee on Psychiatry and Mental Hygiene. The schedule of lecture dates was completed after an exchange of cables between the Salmon Committee and Sir Harold Whittingham, Director General of the Medical Services of the Air Ministry in London.

Dr. Gillespie has received special leave of absence from the RAF from the British government for the express purpose of delivering the Salmon Lectures in this country and Canada. He will fly here to make a first-hand report to members of the American medical profession and officers of the United States Army and Navy Morale Division on the psychological effects of "Blitz" warfare on civilian and armed forces.

The schedule for the Salmon Lectures is as follows

November 17, 18	New York City
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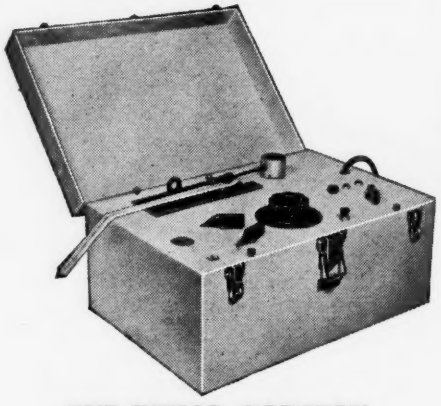

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1 Ebaugh, F. G.: "The Electroencephalogram," Am. J. M. Sc., 196: 882, 1938.

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